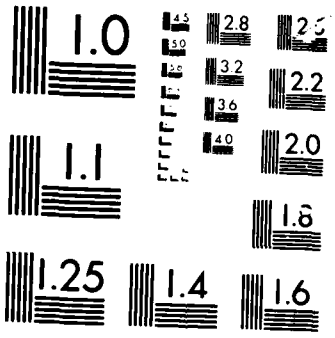
AD-A169 208 QUANTIFICATION OF RESPIRATORY MUSCLE PERFORMANCE(U) 1/1
ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA
S R MUZA JUN 86 USARIEM-M38/86

UNCLASSIFIED F/G 6/16 NL



MICROCOPY

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: M38/86
2. GOVT ACCESSION NO.:
3. RECIPIENT'S CATALOG NUMBER:

4. TITLE (and Subtitle): Quantification of Respiratory Muscle Performance
5. TYPE OF REPORT & PERIOD COVERED:
6. PERFORMING ORG. REPORT NUMBER:

7. AUTHOR(s): Stephen R. Muza
8. CONTRACT OR GRANT NUMBER(s):

9. PERFORMING ORGANIZATION NAME AND ADDRESS: U.S. Army Research Institute of Environmental Medicine, Natick, MA 01760-5007
10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: 3E162777A879, WU 127

11. CONTROLLING OFFICE NAME AND ADDRESS: Same as 9. above
12. REPORT DATE: June 1986
13. NUMBER OF PAGES: 10

14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office):
15. SECURITY CLASS. (of this report): Unclassified
15a. DECLASSIFICATION/DOWNGRADING SCHEDULE:

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution is unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

DTIC ELECTE JUN 27 1986 S D A

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

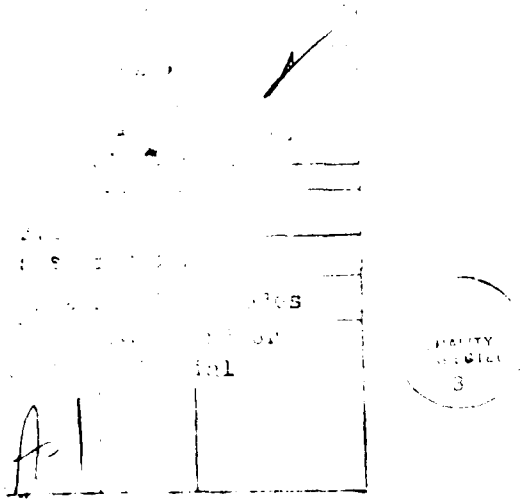
Respiratory muscles, Muscle strength, Muscle performance, Exercise

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Quantification of respiratory muscle force, velocity and length are usually indirect measurements assessed from pressure, air flow and volume, respectively. Pressure produced by the respiratory muscles is measured at sites within the airways, rib cage and abdomen. Quantification of diaphragmatic tension is obtained by measurement of transdiaphragmatic pressure (P_{di}) which is the difference between gastric and esophageal pressures. The strength of the inspiratory muscles working in concert is the voluntary static maximum inspiratory pressure measured at the mouth, briefly sustained near functional residual capacity (FRC).

AD-A169 208
DTIC FILE COPY

Maximal P_{di} is measured during the same maneuver. The inspiratory muscles' velocity of shortening is quantified by measuring the inspiratory flow rate (V_I). Since the V_I varies over the duration of inspiration (T_I), the mean inspiratory flow rate (V_I/T_I) is typically used. Finally, changes in the respiratory muscles' length are inferred from measurements of volume. When the lung volume is at its FRC, the inspiratory muscles are at their resting length, and altering lung volume above or below FRC will shorten or lengthen the muscles respectively. The duration of sustained voluntary hyperpnea is inversely related to the time-pressure integral of the diaphragm ($T_I/T_T \cdot P_{di}\ \bar{P}_{di}$ max) and the mean V_I. Thus, measurements of pressure, flow and volume yield quantitative indices of respiratory muscle performance.



QUANTIFICATION OF RESPIRATORY MUSCLE PERFORMANCE

S. R. Muza

U.S. Army Research Institute of Environmental Medicine

ABSTRACT

Quantification of respiratory muscle force, velocity and length are usually indirect measurements assessed from pressure, air flow and volume, respectively. Pressure produced by the respiratory muscles is measured at sites within the airways, rib cage and abdomen. Quantification of diaphragmatic tension is obtained by measurement of transdiaphragmatic pressure (P_{di}) (gastric minus esophageal pressures). The strength of the inspiratory muscles working in concert is the voluntary static maximum inspiratory pressure measured at the mouth, briefly sustained near functional residual capacity (FRC). The inspiratory muscles' velocity of shortening is quantified by measuring the inspiratory flow rate ($\dot{V}_I$). Since the $\dot{V}_I$ varies over the duration of inspiration (T_I), the mean inspiratory flow rate ($\dot{V}_I/T_I$) is typically used. Finally, changes in the respiratory muscles' length are inferred from measurements of volume. The duration of sustained voluntary hyperpnea is inversely related to the time-pressure integral of the diaphragm ($T_I/T_T \cdot P_{di}/Pdi$ max) and the mean $\dot{V}_I$. Thus, measurements of pressure, flow and volume yield quantitative indices of respiratory muscle performance.

QUANTIFICATION OF RESPIRATORY MUSCLE PERFORMANCE

S. R. Muza

U.S. Army Research Institute of Environmental Medicine

ABSTRACT

Quantification of respiratory muscle force, velocity and length are usually indirect measurements assessed from pressure, air flow and volume, respectively. Pressure produced by the respiratory muscles is measured at sites within the airways, rib cage and abdomen. Quantification of diaphragmatic tension is obtained by measurement of transdiaphragmatic pressure (P_{di}) ~~which is the difference between~~ (gastric minus ~~and~~ esophageal pressures). The strength of the inspiratory muscles working in concert is the voluntary static maximum inspiratory pressure measured at the mouth, briefly sustained near functional residual capacity (FRC). ~~Maximal P_{di} is measured during the same maneuver.~~ The inspiratory muscles' velocity of shortening is quantified by measuring the inspiratory flow rate ($\dot{V}_I$). Since the $\dot{V}_I$ varies over the duration of inspiration (T_I), the mean inspiratory flow rate (V_I/T_I) is typically used. Finally, changes in the respiratory muscles' length are inferred from measurements of volume. ~~When the lung volume is at its FRC, the inspiratory muscles are at their resting length, and altering lung volume above or below FRC will shorten or lengthen the muscles respectively~~. The duration of sustained voluntary hyperpnea is inversely related to the time-pressure integral of the diaphragm ($T_I/T_T \cdot \tilde{P}_{di}/Pdi\ max$) and the mean $\dot{V}_I$. Thus, measurements of pressure, flow and volume yield quantitative indices of respiratory muscle performance.

INTRODUCTION

The quantification of respiratory muscle performance is useful in the study of respiratory physiology. Spontaneous and unassisted ventilation of the lungs is dependent upon neural stimulation and contraction of the inspiratory muscles. Expiration is generally passive with low metabolic activity and no abnormal pulmonary pathology. Inspiratory air flow is generated by many different muscles operating through a variety of complicated lever systems. The geometrical and anatomical complexities of the chest wall (rib cage and abdomen) make the direct measurement of respiratory muscle force, velocity and length impractical in human subjects. However, it is possible to measure the pressures, air flows and volumes generated by the action of the respiratory muscles on the lungs and airways. The purpose of this paper is to provide a brief review of the techniques used to assess respiratory muscle performance in conscious human subjects.

METHODOLOGIES

Measurement of Force

The quantification of respiratory muscle force or tension is indirectly measured from the pressures produced by the respiratory muscles at various sites within the airways, rib cage and abdomen. By convention, pressures measured within the respiratory system are referenced to atmosphere and expressed in cm H_2O (1). The simplest pressure to measure is the pressure at the airway opening commonly referred to as mouth pressure (P_m). The P_m is usually measured in the center chamber of a non-rebreathing valve or mouthpiece (Fig. 1). A short

length of a polyethylene catheter (Intramedic PE-200) connects a differential pressure transducer (Validyne Model MP 45-1-871) to a sample port on the non-rebreathing valve. The range of the differential pressure transducer is varied depending upon the experimental conditions. The amplitude of the fluctuation in P_m during breathing is dependent upon the inspiratory and expiratory flow rates and the resistance of the respiratory apparatus to air flow. Experimental procedures which apply added resistive or elastic loads to the breathing circuit may generate a large range of P_m. Typically, a differential pressure transducer with a range of $\pm$ 50 cm H_2O is used to measure P_m during exercise and rest with small ($R < 10$ cm $H_2O \cdot l^{-1} \cdot s^{-1}$) or moderate ($R < 30$ cm $H_2O \cdot l \cdot^{-1} s^{-1}$) resistive loads, respectively (Fig. 2). The strength of the inspiratory muscles working in concert is the voluntary static maximum inspiratory pressure (P_{Imax}). The P_{Imax} is measured at the mouth during a briefly sustained maximal inspiratory effort against a closed airway at the end of a normal expiration (2). A small port in the mouthpiece ensures that the subject maintains an open glottis during the maneuver. The strength of the expiratory muscles (P_{Emax}) can likewise be assessed by a maximal expiratory effort against a closed airway at the end of a maximal inspiration. During P_{Imax} and P_{Emax} maneuvers, peak pressures may reach $\pm$200-300 cm H_2O. Measurement of the P_{Imax} and/or P_{Emax} before and immediately after a sustained breathing task can provide an index of the degree of respiratory muscle fatigue which may have developed during the ventilatory exercise task. It should be noted that although the P_{Imax} and P_{Emax} measurements are influenced primarily by the strength of the respiratory muscles, passive elastic properties of the respiratory system also affect the achieved pressures. The actual maximal pressures developed by the respiratory muscles (P_{mus}) can be calculated by correcting for the passive elastic forces determined from each subject's relaxation pressure-volume curve (3).

The principal muscle of inspiration is the diaphragm. Quantification of diaphragmatic tension is obtained by measurement of the transdiaphragmatic pressure (P_{di}). The P_{di} is defined as the difference between the abdominal and pleural pressures (4). Abdominal and pleural pressures are usually measured in the stomach and esophagus, respectively. The gastric pressure (P_{ga}) and esophageal pressure (P_{es}) are assumed to be representative of the pressures everywhere in their respective compartments. Usually, the P_{ga} and P_{es} are measured from thin-walled latex balloons sealed on 100 cm lengths of polyethylene catheter (5). The gastric balloon is positioned in the stomach (Fig. 1) and filled with 1.0 ml of air, while the other balloon is positioned in the lower third of the esophagus and filled with 0.5 ml of air. The ends of the polyethylene catheters are connected to two identical differential pressure transducers with identical sensitivities. The difference between P_{ga} and P_{es} (P_{di}) is determined electrically. The frequency response of the balloon-catheter-transducer system is flat to 20 Hz. Recently, I have replaced the balloon-catheter-transducer system with ultraminiature pressure sensors affixed to the distal end of a catheter (Millar Mikro-Tip Catheter Pressure Transducer). Advantages of the catheter pressure transducers include a higher frequency response and a smaller catheter diameter which eases insertion and minimizes subject discomfort. The higher frequency response of the catheter pressure transducer ensures accurate measurement of P_{di} when breathing is opposed by externally added resistive loads and breathing rate is high. Sample tracings of P_{ga} and P_{es} are shown in Fig. 2. Maximal P_{di} (P_{dimax}) can be measured using the same maneuver previously described for measuring P_{Imax}. Some investigators achieve larger P_{dimax} by combining the maximal inspiratory effort with an expulsive maneuver designed to increase the P_{ga} component of the P_{di} (6).

Measurement of Velocity

The quantification of velocity of respiratory muscle shortening is derived from the measurement of the rate of air flow (7). The assumption is that the velocity of shortening of the inspiratory muscles increases with increasing inspiratory flow rate. However, since the inspiratory air flow rate, like respiratory muscle force, is generated by many different muscles functioning in a complex anatomical structure, no clear relationship between the rate of air flow and the velocity of shortening of all the different respiratory muscles has been demonstrated (7). Furthermore, before inspiratory air flow commences, the alveolar gas pressure must be decompressed by inspiratory muscle shortening, which is not a component of the air flow measured at the mouth. Likewise, distortion of the rib cage and abdomen without changes in lung volume require respiratory muscle shortening, which does not generate externally measured air flow. As ventilation and, consequently, the air flow at the mouth increase, that portion of the muscle shortening which does not contribute to the externally measured air flow decreases. Therefore, air flow measured at the mouth probably reflects the velocity of shortening of the inspiratory muscles at elevated levels of ventilation (8). Inspiratory air flow ($\dot{V}_I$) can be measured with a pneumotachograph (Hewlett-Packard 47304A) positioned in line with the inspiratory portion of the breathing circuit (Fig. 1). The pneumotachographs's output can be sampled continuously to attain instantaneous measurements of flow from which the shape of the flow curve and peak flows (Fig. 2) can be obtained. If a spirometer or dry gas meter is utilized to measure ventilation, the mean inspiratory flow rate can be calculated by dividing the cumulative inspired volume by the measured duty cycle (inspiratory duration/breath duration).

Measurement of Length

In the respiratory system, the volume of air ventilated reflects changes in the respiratory muscles' length. During inspiration, the chest wall expands and the inspiratory muscles shorten in proportion to the inspired volume. Likewise, during expiration the chest wall contracts and the expiratory muscles shorten in proportion to the expired volume. Inspired or expired volume is easily measured by spirometry or integration of the air flow. Due to the complex structure of the respiratory system, a given lung volume can be attained with a variety of abdominothoracic (chest wall) configurations. Consequently, it is not possible to relate lung volume measured at the mouth to length changes of specific respiratory muscles.

By measuring the movements and distortions of the chest wall, one can infer the action and activity of specific respiratory muscles and muscle groups (9). The chest wall is divided into two parts: the rib cage (RC) and abdomen (Abd). Konno and Mead (10) established that changes in the anteroposterior diameter of the RC and Abd are closely related to lung volume changes. To measure these linear displacements of the chest wall in humans, magnetometers attached to the anterior and posterior surfaces of the RC and Abd have been used (9). Another index of chest wall displacement is cross-sectional area. A respiratory inductive pneumograph using two elastic bands, applied around the RC and Abd (Fig. 1), can be calibrated to give an output approximately equal to the cross-sectional area within each band (11). Thus, displacement of the entire chest wall can be measured by quantifying the volume of air inspired or expired at the mouth. But measurement of volume displacements (length changes) of the rib cage or abdomen are usually estimated indirectly from measurement of anteroposterior dimensions or cross-sectional area (9).

Measurement of Respiratory Muscle Endurance

By assigning a specific ventilatory ($\dot{V}_E$) target which is characterized by one or more of the following parameters: tidal volume, duration of inspiration and/or expiration and magnitude of pressures required, respiratory muscle endurance can be measured by the ability to sustain the ventilatory target (12). Roussos and Macklem (13) found that the endurance time of the human diaphragm is less than 60 minutes when the P_{di} developed with each inspiration is greater than 40% of the subject's P_{dimax}. Bellemare and Grassino (14) demonstrated that the development of diaphragm fatigue was dependent upon both the relative P_{di} developed and the duration of the contraction or duty cycle (T_I/T_T). This time tension index ($\bar{P}_{di}/P_{dimax} \cdot T_I/T_T$) was found to have a critical value of about 0.15. Above this value, diaphragm fatigue would develop and limit ventilatory endurance time (T_{LIM}) to less than 45 minutes. Recently, McCool et al. (8) demonstrated that at a given time-pressure integral of the inspiratory muscles, T_{LIM} increased as the inspiratory resistance opposing breathing increased and therefore as mean inspiratory flow rate decreased. They concluded that the inspiratory muscle endurance is inversely related to the time-pressure integral and velocity of shortening of the inspiratory muscles.

APPLICATION

The methodologies described here are currently being utilized in our laboratory to evaluate the considerable variability observed between subjects in the degree of discomfort felt and tolerance to exercise when inspiration is opposed by externally added resistive loads. The ventilatory responses to

progressive intensity exercise on a cycle ergometer are plotted for one subject in Fig. 3. As minute ventilation increases, the external respiratory work rate likewise increases. When a 5 cm $H_2O \cdot l^{-1} \cdot s^{-1}$ resistance was added to inspiration, the respiratory work rate was, as expected, greater at any given minute ventilation. The solid line represents the external respiratory work rate at which 50% of the population should experience dyspnea (15). When the subject breathed against the added inspiratory resistance, he was always at or above this level and was unable to achieve the same exercise intensity that he previously attained with minimal inspiratory resistance. This respiratory limitation to exercise imposed by added air flow resistance has been previously described by numerous investigators (15). However, using these methodologies, future studies will evaluate the relationship between a subject's sensitivity to respiratory sensations and their tolerance to exercise when breathing is opposed by resistive loads.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation. Approved for public release; distribution unlimited.

REFERENCES

1. Bartels, H., P. DeJours, R.H. Kellogg and J. Mead. Glossary on respiration and gas exchange. J. Appl. Physiol. 34:549-558, 1973.

2. Black, L. and R. Hyatt. Maximal respiratory pressure: Normal values and relationship to age and sex. Am. Rev. Respir. Dis. 99:696-702, 1969.

3. Rahn, H., A.B. Otis, L.E. Chadwick and W.O. Finn. The pressure volume diagram of the thorax and lung. Am. J. Physiol. 146:161-178, 1946.

4. Agostoni, E. and H. Rahn. Abdominal and thoracic pressures at different lung volumes. J. Appl. Physiol. 15:1087-1092, 1960.

5. Milic-Emili, J., J. Mead, J.M. Turner and E.M. Glauser. Improved technique for estimating pleural pressure from esophageal balloons. J. Appl. Physiol. 19:207-211, 1964.

6. Laporta, D. and A. Grassino. Measurement of maximal Pdi in humans (Abstract). Am. Rev. Respir. Dis. 129, Part 2: A267, 1984.

7. Agostoni, E. and W.O. Fenn. Velocity of muscle shortening as a limiting factor in respiratory air flow. J. Appl. Physiol. 15:349-353,1960.

8. McCool, D.F., D.R. McCann, D.E. Leith and F.G. Hoppin, Jr. Pressure-flow effects on endurance of inspiratory muscles. J. Appl. Physiol. 60:299-303, 1986.

9. Loring, S.H. and E.N. Bruce. Methods for study of the chest wall. In: Handbook of Physiology. Washington, D.C.: Am. Physiol. Soc., 1985, Sect. 3, Vol. III, Chapt. 2.

10. Konno, K. and J. Mead. Measurement of the separate volume changes in rib cage and abdomen during breathing. J. Appl. Physiol. 22:407-422, 1967.

11. Cohn M.A., A.S.V. Rao, M. Broudy, S. Birch, H. Watson, N. Atkins, B. Davis, F.D. Scott and M.A. Sackner. The respiratory inductive plethysmograph: a new non-invasive monitor of respiration. Bull. Europ. Physiopath. Resp. 18:643-658, 1982.

12. Leith, D. and M. Bradley. Ventilatory muscle strength and endurance training. J. Appl. Physiol. 41:508-516, 1976.

13. Roussos, C.S. and P.T. Macklem. Diaphragmatic fatigue in man. J. Appl. Physiol. 43:189-197, 1977.

14. Bellemare, F. and A. Grassino. Effect of pressure and time of contraction on human diaphragm fatigue. J. Appl. Physiol. 53:1190-1195.

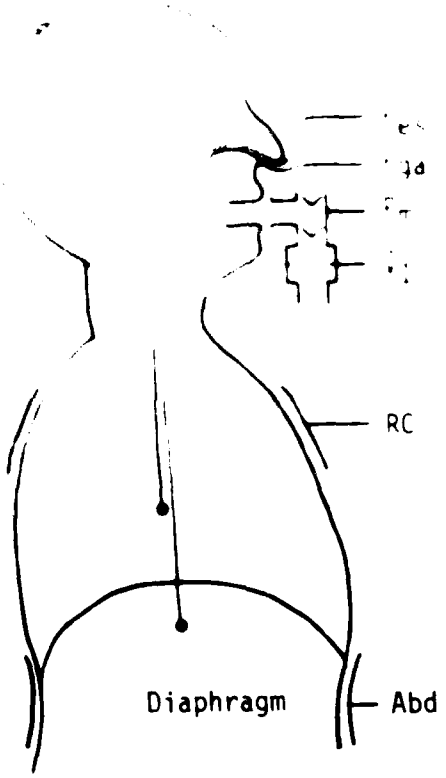
15. Bentley, R.A., O.G. Griffin, R.G. Love, D.C.F. Muir and K.F. Sweetland. Acceptable levels for breathing resistance of respiratory apparatus. Arch. Environ. Health 27:273-280, 1973.

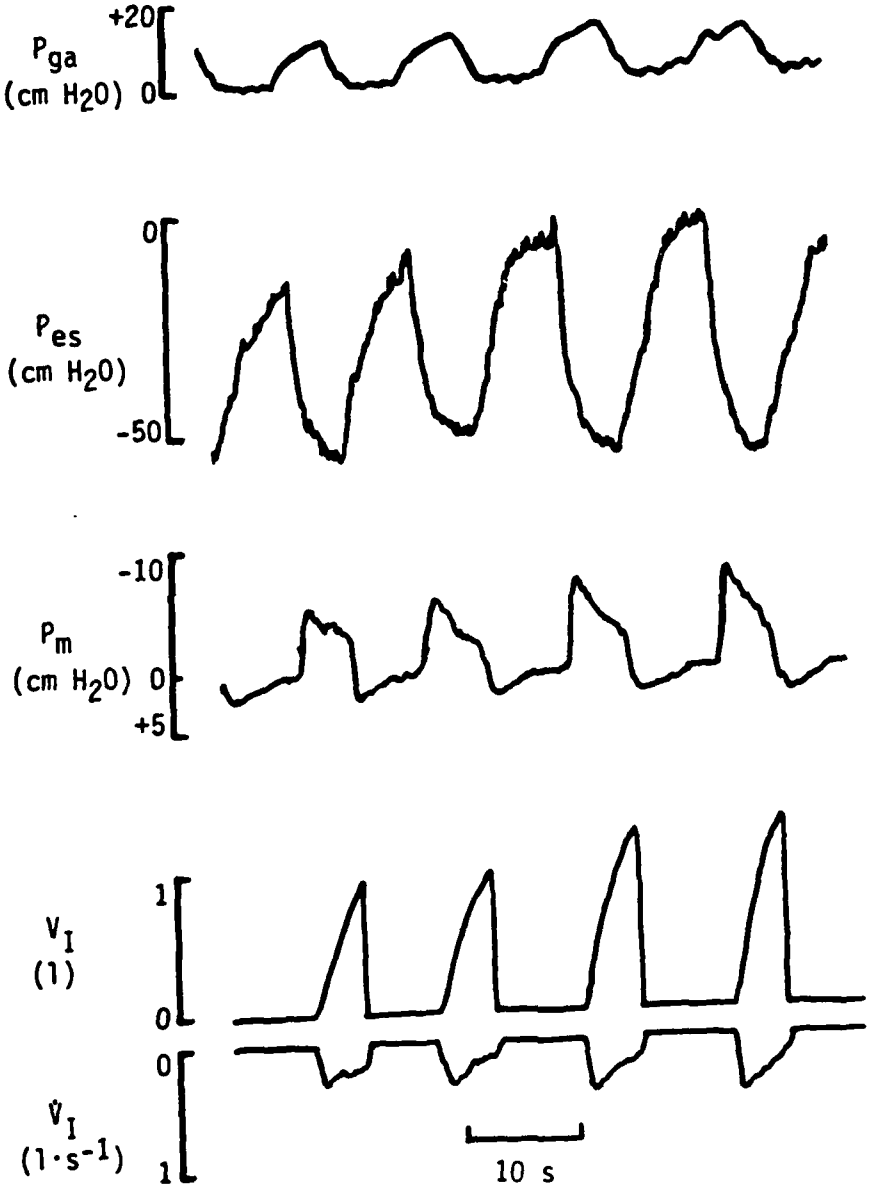
Figure Legends

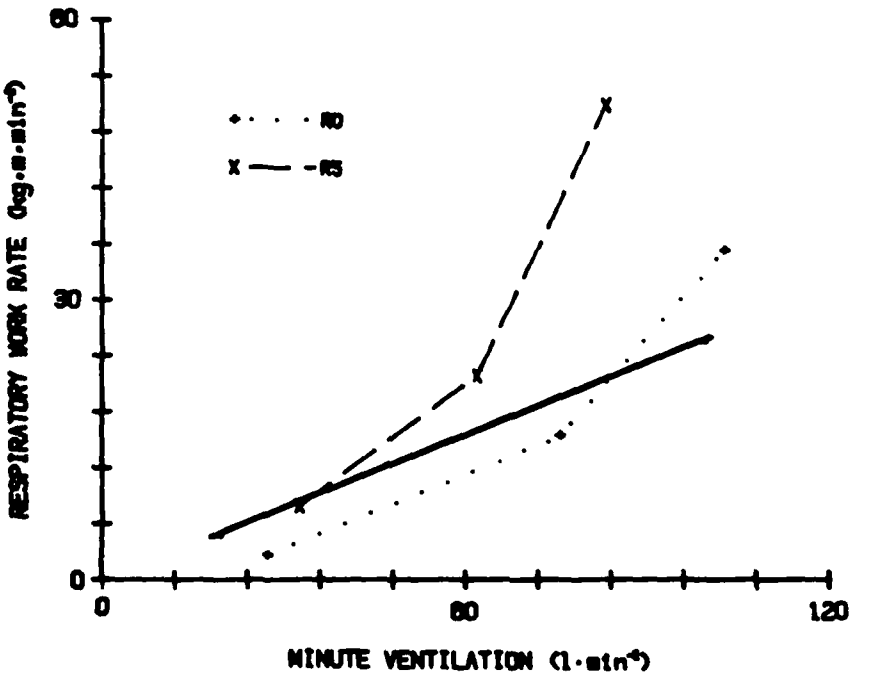
Figure 1: schematic representation of experimental apparatus for measuring force, velocity and displacement of respiratory muscles.

Figure 2: polygraphy record of subject breathing against a 15 cm $H_2O \cdot l^{-1} \cdot s^{-1}$ inspiratory resistance.

Figure 3: respiratory work rate (integral of the instantaneous product of $P_m X \dot{V}_I$) as a function of minute ventilation. RO and R5 are minimal and elevated inspiratory resistance respectively.


RC
Diaphragm
Abd


+20
P_{ga}
(cm H_2O) 0
0
P_{es}
(cm H_2O)
-50
-10
P_m
(cm H_2O) 0
+5
1
V_I
(l)
0
0
$\dot{V}_I$
($l \cdot s^{-1}$)
1
10 s


60
30
0
0
60
120
RESPIRATORY WORK RATE (kg·m·min⁻¹)
MINUTE VENTILATION (l·min⁻¹)

END

DTIC

7-86

END

DTIC

7-86